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VOL. LXIV JANUARY, 1954. No. 1

AIDS TO HEARING.*

KENNETH M. DAY, M.D.,
Pittsburgh, Pa.

The general public has acquired the belief that a hard-of-hearing individual need simply wear a hearing aid to bring his hearing up to normal and thus solve all his hearing problems. This is the idea conveyed by advertisements for hearing aids. Many physicians seem to share this idea. After all, if an individual has defective vision he usually can have it corrected by a properly fitted pair of glasses, and the two problems would seem to be similar. As a result there is a growing public resentment and intolerance towards the hard-of-hearing who do not wear hearing aids. Actually, there is no similarity between the two problems.

A hearing aid cannot be prescribed like a pair of glasses to bring sounds sharply into focus and correct hearing defects. The so-called tailor fitting of a hearing aid is largely a myth. A hearing aid simply amplifies sound like any public address system. It provides loudness or quantity, but not quality. The modern hearing aids do have adjustments that can suppress the amplification of low or high tones to a minor degree, but for the great majority of the hard-of-hearing, uniform amplification throughout the scale provides the best hearing ability. Not all hard-of-hearing persons can wear an electric hearing aid with benefit. This is especially true of traumatic deafness and presbycusis. If there be good hearing

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present for tones below 1,000 cycles but an abrupt nerve loss above that point, amplification of sound may improve the hearing intelligibility for voices but little, if at all.

Modern hearing aids are a boon to mankind and have reached a state of perfection and acceptance where they are providing useful hearing to millions of hard-of-hearing people. With a few exceptions the present vacuum tube aids are pretty well standardized, and the quality of performance of the different makes does not vary much. It would be wise for the purchaser of a hearing aid to ascertain the local facilities for servicing his hearing aid before making a purchase. Otologists should also obtain this information and should refer their patients only to those dealers who can provide repair or replacement service.

With the advent of transistors we can expect further improvements in the hearing aids of the near future. Elimination of the vacuum tubes will enable the aids to be made smaller; there will be no tube noises and it will be possible to increase the amplification of sound without the distortion that occurs with vacuum tubes. There will be a great economy of operation, as transistors require little current and the B batteries of the vacuum tube aids can be eliminated. The transistors are not perfected as yet, and occasionally break down without apparent reason. This is thought to be due to technical faults in their manufacture which are expected to be overcome in the near future.

The manufacturers and distributors of hearing aids are not helping the cause of the hard-of-hearing by their methods of advertising. Do not forget that they are in a competitive business to sell hearing aids and make money. The most effective advertising capitalizes on the psychologic weaknesses of the consumers. Much more stress is put on the size and invisibility of the hearing aids they sell than on the quality and fidelity of response of their instruments. They outdo one another in superlatives and in false implications. The Subcommittee on Audiometers and Hearings Aids in the A.M.A. has attempted to police this advertising with only indifferent success, and Better Business Bureaus are also helping in this re-

spect. The final solution of misleading and improper advertising can only be reached when the hearing aid industry organizes to police itself. This it proposes doing, and we await results.

The major problem faced by those with defective hearing is the ability to converse with their fellow men. This paper is primarily concerned with the means of improving that ability. Electric hearing aids provide only loudness. The best hearing aid is one which provides this loudness with the least distortion. For those persons with a pure conductive type of deafness, loudness is all that is needed to enable them to hear distinctly. For those with mixed or nerve types of deafness mere loudness is not enough to provide distinct hearing. The greater the cochlear or nerve damage for tones within the speech range the less will be the power of discrimination for speech; moreover, because of the recruitment factor, too much loudness itself will reduce the power of discrimination. This is the reason why so many of the hard-of-hearing persons have difficulty in understanding conversation in the presence of much background noise.

I have a message to deliver to the hard-of-hearing and to those who deal with them as doctors or teachers, which might be called a philosophy for the deafened. In order to make my remarks authoritative and to explain this philosophy it will be necessary for me to relate some of my own experiences. I present my case history with no feeling of braggadocio, but merely in an attempt to prove what can be accomplished in the art of communication by people with serious hearing difficulties if they have the determination to overcome those difficulties and the willingness to make needed adjustments.

I have otosclerosis of the semi-malignant type with early cochlear involvement. There is no record of otosclerosis on either side of my family. My deafness first became apparent while I attended college, and increased rapidly. In 1917, after being discharged from the army because of my deafness, I entered medical school. Although I managed to complete the medical courses satisfactorily, my medical degree was temporarily withheld for the ostensible reason that my deafness

might adversely affect my medical career and cause discredit to the institution. This was quite a shock to me, and marked the beginning of a period of depression and despondency which continued for some ten years. During this period I studied lip-reading, but I never became a proficient lip-reader. I also submitted to various operations and treatments which supposedly might improve my hearing. Surprisingly, I did seem to hear better following some of these procedures. Later I discovered that the apparently improved hearing was simply improved attention. Between times I had fallen into a common habit of the hard-of-hearing of not listening and I, therefore, seemed deafer than I actually was. I acquired a reputation for being a snob, because I often failed to reply to people who spoke to me. The medical practice which I had largely inherited from my father dwindled rapidly, and I became a subject of serious concern to my family. It was during this period that the carbon type of hearing aid appeared on the market, and I half-heartedly tried wearing one. I did not like it. Though I admittedly heard better, the distortion of sound and extraneous noises were very bothersome, and even with the hearing aid, I could understand only about one-half of what I heard; moreover, I felt conspicuous, and it was a severe blow to my pride to be advertising an infirmity which I had been trying to conceal for years.

A former patient of mine who proved to be a real friend did more to bring me to my senses than any other person. He told me that the reason he and other patients had left me and consulted other doctors was because of my attitude of indifference when they tried to tell me about their complaints; that I acted as though I were not listening to them, and as a result they could not determine whether or not I understood them. Consequently they were afraid that I would treat them improperly. This was quite true. I had thought that I was carrying on a successful bluff by pretending to hear what they said, whereas I actually was not even trying to listen to them half the time. I was rudely awakened to the fact that in trying to conceal my deafness I was fooling no one but

myself; moreover, I was beginning to realize that my hearing was not going to improve with the help of any miracle treatments, but was actually growing gradually worse.

It was at this stage of my life that I finally accepted my deafness as an unalterable fact, and acquired the determination to overcome my handicap. I began to wear a hearing aid continuously, but without my former feeling of antagonism towards it. As I became used to the distortion and extraneous noises I found that my discrimination improved. I also noticed that if I wore the aid conspicuously people would speak to me a little more slowly and distinctly, and that fact alone improved my understanding for speech.

I began to use my eyes and my mind to help me listen and became increasingly adept as a speech reader, though I never did master the art of lipreading. If I can see the facial expression and catch some of the accented syllables, I can usually follow the trend of a conversation without much difficulty with the help of what sound I can hear through my hearing aid. I discovered that by wiring my hearing aid with a receiver for each ear I could understand much more than I could with a receiver in only one ear. This procedure, however, has its drawback. With both ears plugged by receivers it is very difficult to modulate one's own voice. It took nearly two years before I could control the modulation of my voice satisfactorily. This is probably the sole reason that so few individuals will wear a hearing aid with receivers in both ears, though I consider it a small price to pay for better hearing. When I became adjusted to my own disability my otologic practice increased rapidly, and many of my former patients returned to me. I soon discovered that my deafness, instead of being a handicap, was actually one of the biggest assets to my practice. Many deafened people consulted me because they believed I could understand their problems better since I also was hard-of-hearing.

I believe that one of the surest signs of the acceptance of and adjustment to a handicap or infirmity is the development of a sense of humor about that handicap. I, personally, take great delight from intentional misinterpretations of remarks

made to me. A bizarre example of this occurred recently when I insisted that someone said that I was a nice, old bachelor when what he actually said was that I was a dirty, old bastard.

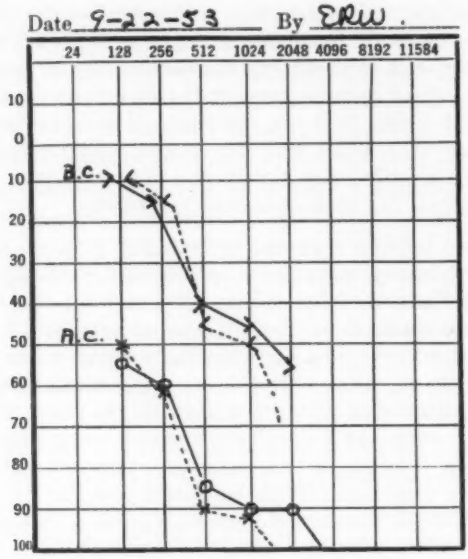

Fig. 1.

With the advent of vacuum tube hearing aids the quality of sound reproduction improved considerably, and much of the distortion which was produced by the carbon type of aids was eliminated. Although my hearing loss has increased somewhat in recent years I can understand more today with the present aids than I could 15 years ago. I believe that further improvement in quality will be attained with the transistor aids, aside from the reduction in their size and weight.

To complete my case history I am presenting a copy of my audiogram made just a few weeks ago by the Audiology Department of the University of Pittsburgh. (Fig. 1). Discrimination tests revealed that I have a 20 per cent discrimination

score for my left ear and a 30 per cent score for my right ear for recorded voice with amplification of 94 decibels in a soundproof room. Using receivers on both ears my discrimination improved to 48 per cent. Greater amplification caused a sharp drop in discrimination. From these records it can be seen readily why I wear receivers in both ears.

My experience is not an unusual one, but is simply an example of thousands of similar cases, though my deafness is more severe than most. I have seen many a household disrupted because one hard-of-hearing individual would make no attempt to adjust to his deafness or accept any of the aids available for better hearing. This often has been due to lack of proper guidance or advice by his physician. In this modern era with the facilities available for helping the deafened there is little excuse for such an occurrence.

As a result of my own experience I have a few words of advice to give to the otologists. If you find that medical or surgical treatment is ineffective, do not continue to treat your deafened patients or give them hope that their hearing may improve. They will make no attempt to adjust to their handicap so long as they have any hope of improved hearing from treatment. Fully one-half of the maladjusted deafened people today have failed to make any attempt to help themselves, because physicians continue to hold out straws of hope for the restoration of hearing by means of miracles. It is an unpleasant fact that the great majority of our deafened patients cannot be helped by treatment, and we do them serious harm by continuing to treat them either in the hope of improving their hearing or with the false idea of preventing their hearing loss from growing worse. The longer that false hopes are extended to these people and necessary adjustments delayed the more difficult does it become to make these adjustments. On the other hand you will do these patients just as much harm by telling them that their hearing will get worse. The worst offense of all is to intimate that a patient may eventually become stone deaf.

Do not brush these patients off with the suggestion that they investigate hearing aids. They need advice and guidance,

and it is the duty of the otologist to give it to them. Discuss their problems with them. Explain the use and limitations of hearing aids. Refer them to the local audiology department or rehabilitation center. Urge them to join the local league of the American Hearing Society, where they will meet other people more deafened than they are. There they will see for themselves how happy and cheerful the hard-of-hearing can be, and how well they converse after proper training. They can also get instruction in the use of a hearing aid and in speech and auditory training when indicated. The rapid development and growth of auditory centers in this country is an indication of the present recognition of the need for this type of work.

Finally I have a message for the severely deafened which I present as a philosophy for the hard-of-hearing.

Accept your deafness as an incontrovertible fact and acquire the determination to overcome the disability that it causes. Half way measures will not work. Complete acceptance is necessary before needed adjustments can be made.

Get yourself a hearing aid and wear it continuously as a part of your clothing. Do not try to conceal your hearing aid or the receiver in your ear. Discover how much it helps you to have people with whom you converse know that your hearing is defective. The average American is a courteous individual, and he will speak a little more slowly and distinctly if he sees that you are hard-of-hearing.

Be attentive and use your eyes and brain to help you listen. If your discriminative power is poor, you should study lip reading and get auditory training to help improve your hearing ability. You should wear receivers in both ears if by so doing your discrimination is improved, even though this entails the added problem of learning to control the modulation of your own voice.

Develop a sense of humor about your deafness. You will be surprised to learn that if you can laugh at yourself other people will laugh with you rather than at you.

Avoid situations where acute hearing is essential and you will be made to feel conspicuous. Take up a hobby to keep you busy and contented, and to replace activities where acute hearing is needed. Keep your mind actively occupied. A busy mind finds no time for brooding or self-pity.

Avoid the anti-social tendency of the hard-of-hearing. Get out and mingle with your friends and acquaintances and discover how ready they are to help you if you will only help yourself.

If you do these things you may be surprised to find that you are not really handicapped after all, and may thus attain peace of mind and tranquillity of spirit.

OCCLUSION OF THE ROUND WINDOW BY OTOSCLEROSIS.*†

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Occlusion of the round window by otosclerosis has become of clinical interest since the development of fenestration surgery. Occlusion of the round window along with stapes ankylosis has been found on histopathologic examination in a small number of cases, all of which were known to be profoundly deaf. Histopathologic proof that occlusion of the round window may occur without stapes and ankylosis has not been reported. The principal case to be described in this report demonstrates almost total occlusion of the round window without stapes ankylosis or involvement of the oval window region.

In cases of otosclerosis with the usual indications of stapes fixation and good cochlear function, the creation of a new fenestra over the ampulla of the horizontal semicircular canal has as a general rule allowed improvement in hearing up to approximately the 25 db. threshold level. An exception to this rule has been the occasional case in which, notwithstanding good bone conduction and other evidence of normal cochlear function, the hearing has failed to improve following the creation of a technically satisfactory window.

One such case history is briefly presented below. Preoperative and postoperative audiograms are illustrated in Fig. 1.

A. G., 398079. This white man, age 31, was first seen on Jan. 4, 1947. He complained of a gradual loss of hearing in both ears since the age of 16. A diagnosis of otosclerosis was made and a fenestration was done on the left ear on February 7, 1947. There were no operative or post operative complications. Although the fistula response has remained strongly active there has never been any appreciable improvement in hearing at any time after operation.

* Presented before the Otosclerosis Study Group Division of the American Academy of Ophthalmology and Otolaryngoloy, October 11, 1953.

† From the Division of Otolaryngology of the University of Chicago.

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Cases of this type have been observed by various fenestration surgeons and were discussed by members of the Otosclerosis Study Group in 1950.[1,2] Nilsson,[3] and Skoog[4] recently reported two such cases in a series of 53 fenestrations during which they performed accurate audiometric examinations at various stages of the operation.

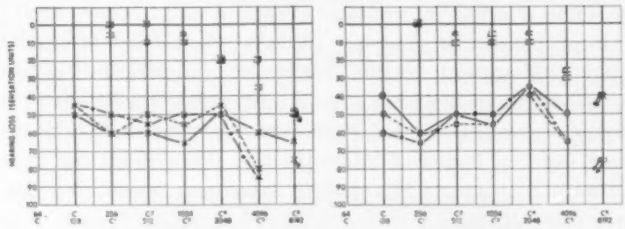

Fig. 1. Bilateral otosclerosis. Male, age 31. Fenestration, left ear. February 7, 1947. Pure tone air and bone conduction thresholds are shown for both ears preoperatively, six weeks postoperatively and six months postoperatively. Note that the postoperative thresholds in the operated ear showed no significant improvement at any time.

A probable explanation for failure to obtain an improvement in hearing in this type of case would be a total occlusion of the round window with a normally functioning oval window. Histopathologic proof has not yet been reported. Such an occlusion obviously could not be differentiated from stapes fixation by the routine tests now employed.

PHYSIOLOGICAL CONSIDERATION.

It is well established that the principal pathway through which sound enters the inner ear is by way of the oval window, and that the round window functions to permit mobilization of the incompressible cochlear fluids; however, it is evident that some sound must reach the round window through the air in the tympanic cavity. This would be in opposite phase to the normal stimulus. Lawerence,[5] however, has shown that this interference is negligible (about .3 db.). Under abnormal conditions the cochlea can be stimulated via the round window. Bekesy[6] has shown that after a radical mastoid op-

eration, the sound may excite the cochlea through the round window route. Likewise several workers (Wever, Lawerence, and Smith;[7] Kobrak;[8] Kobrak, Lindsay and Perlman[9] have demonstrated cochlear stimulation by way of the round window in experimental animals.

Studies have been made on the effect on hearing of blocking the round window in animals. Hughson and Crowe[10] reported on improvement in hearing in cats when they blocked the round window with cotton. Wever and Lawerence[11] reported that all forms of blocking caused only minor variations in sensitivity; however, in a later series of experiments Wever[12] produced hearing losses of 25-30 db and interpreted the failure to obtain such losses in previous experiments as being due to incomplete occlusion. Wever stated that an air bubble of only .3 cubic mm. against the round window membrane would be effective in mobilizing the perilymph for normal cochlear function.

Measurements of bone conduction thresholds in the presence of an adequately blocked round window and a patent oval window have not been reported, as far as we know.

OTOSCLEROSIS OF THE ROUND WINDOW REGION.

The round window area is a common site for the occurrence of an otosclerotic lesion. Guild[13,14] states that "the round window region is next to the oval window region in number of otosclerotic areas. In 113 foci from 81 otosclerotic ears there were 25 lesions involving some part of the attachment of the round window membrane. Thus about one-fifth of all otosclerotic areas in his material were somewhere in this area. Guild further states that "only rarely do areas in this region develop into major lesions, i.e. into those that occlude the round window niche, or that otherwise might affect the acuity of hearing. A review of the chart of Guild's cases reveals that the otosclerotis process was confined solely to the round window area in only two of these ears.

Nylen[15] reporting on 121 otosclerotic ears found that 40 per cent of the foci involved the locality of the round window. In 8 per cent of the ears it was present in only the round window region.

Nager and Meyer[16] report that the round window is second to the oval window in frequency of otosclerotic lesions. In three of 53 ears the round window was the only area involved.

Nager and Fraser[17] published six interesting cases of extensive otosclerosis associated with otitis interna ossificans of the scala tympani. In three of these cases both windows were entirely obstructed. All cases had been profoundly deaf, the

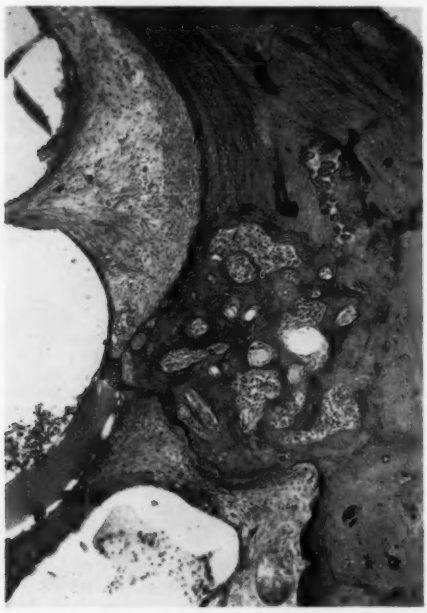

Fig. 2. Photomicrograph of a section through the attachment of the round window membrane and the spiral ligament, showing a minute focus of otosclerosis at the lateral attachment of the membrane. There is no encroachment on the round window. (RWM: Round window membrane.)

This case previously reported in *Archives of Otolaryngology*, Vol. 52, Dec. 1950.

explanation for which was not entirely clear since both degeneration of inner ear structures and occlusion of both windows came into consideration.

We have in our collection of temporal bones at the University of Chicago 37 ears which show otosclerotic lesions. Seventeen of these ears have foci in the round window area. In four ears from three cases the process is exclusively confined to this region. There is a small circumscribed active focus of otosclerosis situated at the lateral attachment of the round window membrane in three of these ears (Fig. 2) obtained from two cases. These foci undoubtedly produced no loss of hearing.

The one ear which was obtained from the third case showed a single focus of otosclerosis occluding nearly all of the round window and will be presented in some detail.

Mrs. B. B., 262696, a 35-year-old white professional singer was under the care of Dr. Ruth E. Taylor of Chicago from 1944 to 1951. When first seen she complained of recurring attacks of pain down the right arm and left chest present since childhood, but worse since a pregnancy in 1941. Nodules were noted about the tendon attachments on the hands and feet. Later she developed substernal pain on exertion, and clinical evidence of arteriosclerotic heart disease. Family history revealed that her mother had died from cardiac disease and that her three brothers suffered from a similar condition.

Blood lipid studies were done on Mrs. B. at the University of Chicago in 1949. The blood cholesterol was 426 mgms. per cent and the total blood lipids were 1155 mgms. per cent. A diagnosis of familial xanthomatosis was made. The same diagnosis has been confirmed in the cases of the three brothers, two of whom have since died of coronary occlusion, while the third has presented symptoms of coronary insufficiency.

During 1947 the patient was seen several times because of a vocal nodule. There were no complaints or abnormal findings referable to the ears. She was aware of the nature of her disease and alert to any new symptoms. She was under close observation by her physician up to the time of her death, but at no time mentioned any auditory symptoms.

On December 24, 1951, she died suddenly when walking in a heavy snow in a blizzard. An autopsy was performed on December 25, 1951, by Dr. Eleanor Humphreys. The findings may be summarized as follows: Familial xanthomatosis tuberosa with xanthofibrous nodules and cholesterol granulomas on the extremities. Severe arteriosclerosis of the aorta and coronary arteries. Multiple scars of small infarcts and extensive patchy fibrosis in the myocardium. The cause of death was acute coronary insufficiency.

The right temporal bone was removed and presented to the Division of Otolaryngology for histopathologic study. The bone was fixed and decalcified in the usual manner. Serial horizontal sections were made 20 microns in thickness.

Histopathology. See Figs. 3 to 6.

Middle Ear. The tympanic membrane, ossicles and mucous membrane were normal. Pneumatization was normal.

Inner Ear. The membranous structures were in a fair state of preservation, but showed moderate degree of postmortem degeneration.

Cochlea. The cochlear duct was normal except for moderate post-mortem degeneration of the organ of Corti. The spiral ganglion and its axons and dendrites were normal. The cochlear nerve was normal. The vestibule, the utricle, saccule and semicircular canals were within normal limits.

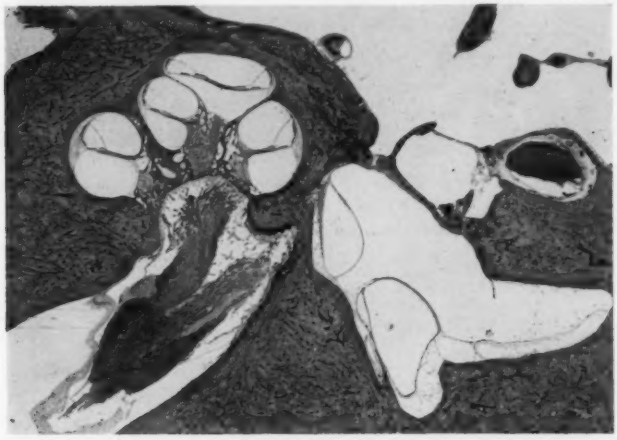

Fig. 3. Horizontal section through the cochlea and vestibule at the level of the oval window. There was no otosclerotic lesion at the site of predilection, or any part of the otic capsule other than that shown in Fig. 4.

Otic Capsule. The periosteal, enchondral and endosteal bone was normal except for an area of typical otosclerosis in the round window area. There was no evidence of otosclerosis in the site of predilection at the oval window or elsewhere in the otic capsule.

The lesion occupied nearly the full thickness of the capsule below the spiral ligament. The round window was completely obstructed in its superior part. A small medial portion remained free, and connected inferiorly by a narrow channel to the middle ear cavity. The mucous membrane adjacent to the focus was thickened and contained many dilated blood vessels, some of which connected directly with vessels in the otosclerotic

focus. This hyperemic mucous membrane further obstructed the open portion of the niche. There was a fracture extending from the posterior edge of the focus to the posterior bony semicircular canal beneath the crista ampullaris, a region in which a small fracture is frequently seen in temporal bones.

A graphic reconstruction of the round window region was made in order to estimate the area occluded by the focus. The total area of the round window was found to be 1.9 sq. mm. The area of the membrane

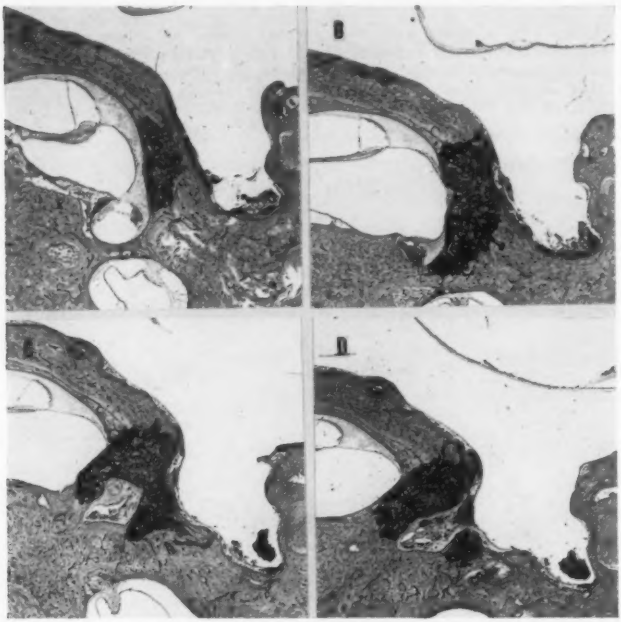

Fig. 4-A. Horizontal section through the basal coil, through the upper aspect of the otosclerotic lesion which lies on the lower promontory adjacent to the spiral ligament at this level.

Fig. 4-B. Section lower than 4-A. The focus occupied almost the full thickness of the capsule. The round window membrane is seen adherent to the otosclerotic focus, except at its medial attachment.

Fig. 4-C. Section below 4-B showing complete occlusion of the round window area at this level. A small portion of the niche remains patent.

Fig. 4-D. Still lower section showing the small remaining communication of the niche within the middle ear.

which was free from otosclerotic bone measured 0.38 sq. mm. Mucous membrane containing many congested vessels, further reduced the remaining air space in the niche. The measurements showed that at least 80 per cent of the round window was obstructed by the otosclerotic bone.

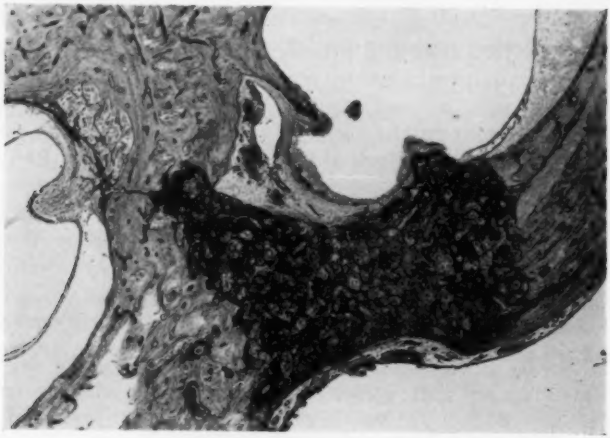

Fig. 5. Higher magnification (x 32) of the lesion. The round window membrane is adherent to the focus anteriorly, but at this level it is free posteriorly. The mucous membrane on both sides of the focus is considerably thickened and contains many dilated and congested blood vessels.

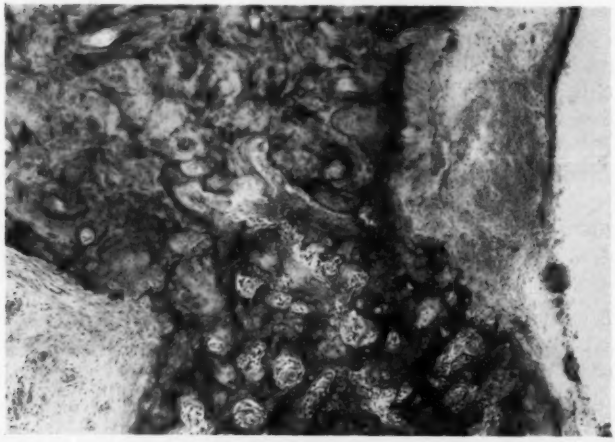

Fig. 6. High power (x 135) showing the typical histopathological appearance of the otosclerotic lesion. The superior half appears sclerotic and inactive while the inferior portion shows many dilated vessels indicating activity.

DISCUSSION.

The case just described provides histologic proof that occlusion of the round window by otosclerosis without involvement of the oval window may occur. The extent and the degree of activity apparent in the focus in this case leaves little doubt that it would have progressed to complete occlusion, had the patient lived. This patient can safely be assumed to have had good hearing until the time of her death. The absence of hearing loss corresponds with the observations made by Wever, who stated that if a small area of the round window membrane remained free the hearing would not be affected. In this case 0.38 sq. mm., equal to 20 per cent of the total area, was free from the otosclerotic focus.

Wever found that occlusion of the round window could cause air conduction loss comparable to that produced by stapes ankylosis. It seems probable that bone conduction thresholds would not be raised correspondingly, although experimental evidence on this point is not available.

If occlusion of the round window is capable of producing air and bone conduction thresholds similar to those found as a result of stapes fixation some method for differentiating the two conditions is needed. Theoretically the principle involved in the Gelle test offers a possible approach, and with modifications to provide for greater precision of measurements might prove to be of practical value. The mobility of the stapes, as determined by testing with the probe during the fenestration operation, may not be a sufficiently accurate indicator of the efficiency of the mechanism as a sound conductor.

SUMMARY.

A case of otosclerosis has been presented in which the focus has been limited to the round window region and has produced occlusion of 80 per cent of the round window membrane.

The absence of hearing loss in this case corresponded to experimental observations made by Wever on the degree of occlusion of the round window necessary to cause hearing loss.

The histologic evidence of activity in this focus suggests that complete occlusion would have eventually developed.

This case confirms the possibility of occlusion of the round window by otosclerosis in the presence of a patent oval window.

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BONE GRAFT FOR ATROPHIC RHINITIS.

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The purpose of this work has been to devise a surgical technique which would tend to restore "normal" nasal function in patients with atrophic rhinitis.

The etiology of atrophic rhinitis has remained obscure. Medical treatment with vitamins, antibiotics, hormone sprays and other drugs has not only failed to improve the condition but even to halt its progress. The surgical management of this condition, however, has been more promising.

Nearly all surgical procedures devised have been directed towards narrowing the functioning airway. It has not yet been found possible to reverse permanently, even in part, the insidiously advancing chronic inflammatory reaction that involves the nasal mucous membranes and the cartilage and bone of the nose, both internally and externally.

Gersuny[1] injected paraffin submucosally, but his results were poor, due to extrusion and spread of the material. Lautenschlager[2] approached the lateral nasal wall via the Caldwell-Luc incision and mobilized this wall medially. The success of the operation depended on synechia between the septum and the turbinates to narrow the air space. Halle[3] modified Lautenschlager's technique, as did Hinsberg.[4] All of these operations, however, interfered with nasal physiology.

Aubonne[5] suggested the use of cartilage and bone implanted subperichondrially in the septum in an effort to narrow the airway. Eisenstodt[6] also suggested the use of cartilage subperichondrially. Beck, quoted by Ersner and Alexander,[7] sug-

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gested the use of bone splinters from the patient's rib, which were placed under the perichondrium of the nasal septum. Zorzoli[8] recently used bovine cartilage in the same way.

Osteotomy of the nasal bone followed by narrowing of the nasal pyramid has been suggested by Ersner and Alexander[7] and Kopp.[9] Narrowing of the nostril and movement of the lateral mucosal wall, together with the inferior turbinate toward the midline, has been suggested by Cottle[10] as another facet of the design for improvement.

Proud,[11] Guttierez,[12] and Causse[13] reported the use of acrylic resins and other plastics in the septal space as a satisfactory method for narrowing the nasal chambers. Kasnetz[14] and Kemler[15] implanted ivory in the same way.

The use of acrylic resins as septal implants has the advantage of making a relatively simple procedure out of the operation. With the Proud technique, it has produced improvement in a number of patients; however, one fears that the pressure of an inert substance will adversely affect the nutrition, and then cause further damage to the nasal mucosa. Inert substances, such as acrylic resin, are also likely to be extruded. Once the mucous membranes have healed following extrusion, dense scarring impairs any future chance to insert another substance. The danger of extrusion also exists with substances such as glass, ivory or metal.

Cartilage and bone used as implants have failed in the past because isogenous or autogenous bone has either been absorbed if tolerated or has been extruded when placed under the perichondrium adjacent to septal cartilage. Resorption rather than extrusion of the cancellous bone took place even when most of the septum has been resected. Cortical bone also behaved in the same way. Cottle[10] has previously used cancellous bone into the septal space and palatal bed area.

In the past, failure of bone grafts to "take" has led to the method's being discarded. A necessary prerequisite of a successful "take" is an adequately prepared bone bed. Atrophic rhinitis involves not only the mucosa and submucosa, but the

underlying bone as well; therefore, an adequate blood supply for the bone graft was difficult to obtain. It was felt, however, that if a bed could be prepared in the palatine bone, the blood supply would be better and a "take" more likely to be secured. If a successful "take" of a cancellous bone graft to the palatine bed could be accomplished, it was reasoned that this graft could assist in the nourishment of the septal mucosa and a more normally functioning mucous membrane. Since a good deal of the septal mucosa is nourished by mucoperiosteum, I have attempted to evaluate the effect of a cancellous bone graft placed in the septum on a bed formed by the palate bones in narrowing the airway and of producing an improved blood supply to the mucoperiosteum of the septum. To exclude other factors which might influence the result, moving the lateral nasal wall towards the midline, narrowing the nasal pyramid or the nares was not done.

Surgical Technique: The operation is divided into two parts: the first consists of securing a cancellous bone graft. The largest piece of cancellous bone available is from the iliac crest. Accordingly a large segment measuring at least three by six centimeters including the entire cortical thickness is removed. All cortical bone is removed from the specimen secured and only the cancellous portion is used. I have facilitated the operative procedure by having another surgeon perform this part of the operation at the same time that the nasal part of the operation is being done.

The second procedure is the operation on the nasal septum. An incision is made over the caudal end of the septum and is carried down onto the floor of the nose and laterally into the ala in order to facilitate admission of the highest and longest graft possible. The caudal part of the septal cartilage is left, but an extensive septal resection is performed leaving only a bar of cartilage under the lateral cartilage. Then using a four millimeter chisel, a deep trough is cut into the hard palate from the anterior nasal spine backwards. The mucous membranes are then separated for a distance along the floor immediately adjacent to the trough, so that no tension will exert on the mucous membrane flaps when the graft is inserted. The graft is then placed in the trough.

The graft is made wide enough that the septal mucous membrane flaps on each side touch the inferior and middle turbinates. The incision is closed with silk. No packing is used.

Following the operation the patient is placed on antibiotics and given crutches to get about. Usually the patient can get about fairly well without the aid of crutches in a week.

The following cases are presented to indicate the results obtained by this procedure:

Case 1: S. R., 51, white, female. Diagnosis: Primary Atrophic Rhinitis with Ozena. Acrylic implant was done by the Proud technique in 1950. There was some symptomatic improvement. Anosmia with minimal crusting remained, and there was no objective change in the nasal mucosa. The

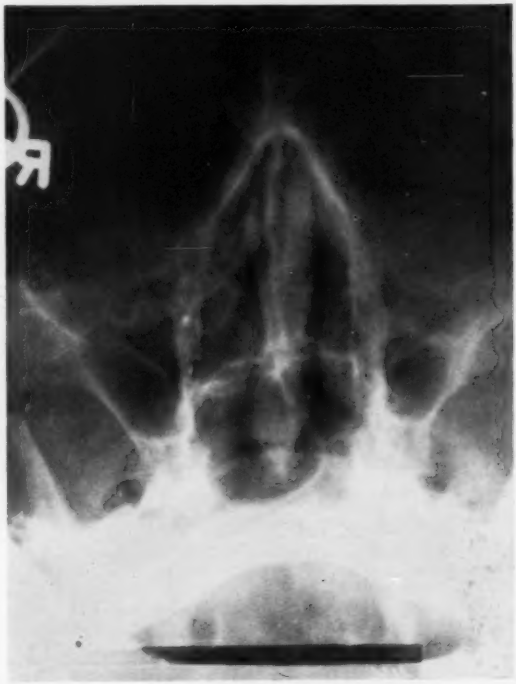

Fig. 1. Two years after surgery. Note the delicate bony trabecular and height of the graft.

patient attributed a weighty feeling in her head to the implant. The implant was removed six months after its insertion and a large single piece of cancellous bone from the hip was placed into the septum in contact with a freshly prepared trough in the bony palate. The septum was widened nearly to the point of objective nasal obstruction, and the patient complained of nasal obstruction for the first three months post operatively.

At present, the nasal mucosa has a normal pink appearance. This change has affected the mucosa over the turbinates as well as that of the septum. The middle and inferior turbinates appeared larger and pinker. Several otolaryngologists who have seen this case without know-

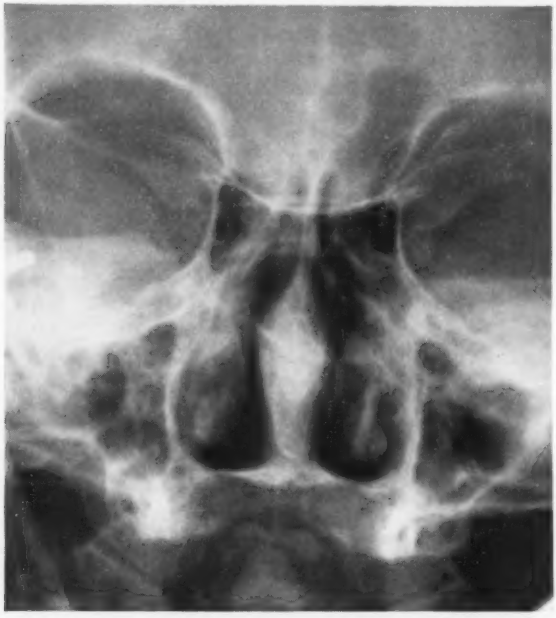

Fig. 2. Two years postoperative. The fusion of bone graft with palate is distinctly visible.

ing about the procedure have remarked that she needs a "submucous resection." The patient remarks that "I can breathe better than at any time in my life and now I can smell my food." Figs. 1 and 2 were taken two years after surgery. The six-months' X-ray showed the graft to be very slightly larger than the present, indicating that some resorption had taken place later.

Case 2: K. M., 17, white, female. Diagnosis: Atrophic Rhinitis and Bilateral Chronic Maxillary Sinusitis. Preliminary bilateral Caldwell-Luc and transantral ethmoidectomy was done when conservative treatment

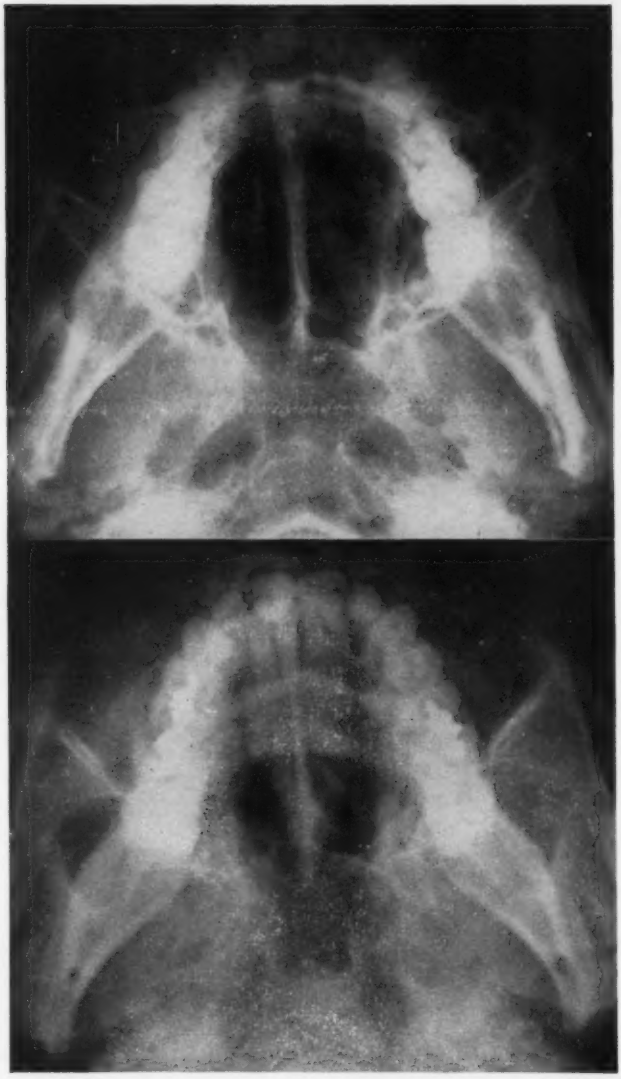

Fig. 3. Submentovertical position. The upper view is a preoperative picture. The lower view, taken two years after surgery, shows a large healthy graft extending posteriorly to the level of the front face of the sphenoid.

failed to improve the condition. One month later a piece of iliac crest was removed. At the time of surgery, it was noted that the cancellous bone was thin and not well developed, and portions of it appeared to be cartilaginous. A single large piece could not be obtained, and even with the insertion of several small pieces of cancellous bone, nasal obstruction could not be obtained. At two months the airway looked ideal, but at three months, it appeared more open and the turbinates remained small and atrophic. On comparison with the previous case this indicated that insufficient bone was placed to narrow the airway sufficiently and with resorption the space became too open. At one year, symptomatically, there was only fair improvement, but objectively there was no change in the appearance of the nasal mucosa.

Case 3: G. C., 58, white, female. Diagnosis: Primary Atrophic Rhinitis and Cylindrical Bronchiectasis. At the time of the removal of the piece from the iliac crest, the cancellous bone was noted to be thin, scant and

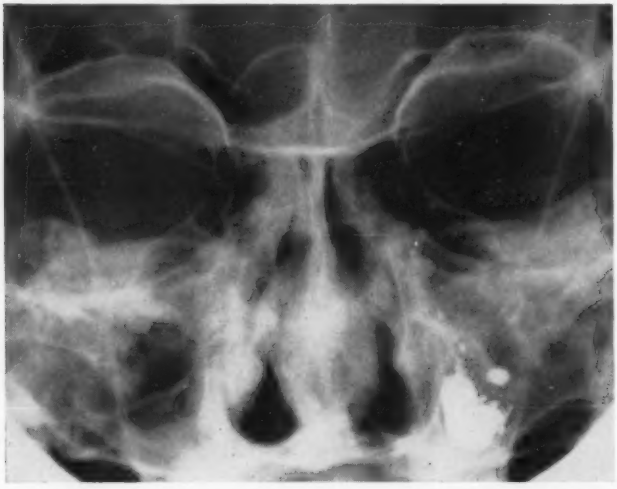

Fig. 4. The graft is a good deal wider at the middle turbinate meatus area.

osteoporotic. Sufficient bone to occlude the airway could not be obtained, although a satisfactory bone bed could be made in the hard palate. Resorption proceeded as rapidly in this instance as in case 2. Symptomatically, she had fair improvement, but objectively there was no significant change.

Case 4: B. R., 29, white, male. Diagnosis: Primary Atrophic Rhinitis. Cyst of right maxillary sinus. The cancellous bone was excellent, and a piece wide enough to obstruct the nasal chambers was placed in the septum. It took fully four months before the nasal chamber opened sufficiently to produce subjectively normal nasal respiration.

It is now two years post-operative (see Figs. 3 and 4). The patient states that he can breathe very well and has no sense of obstruction. Crusting is absent. The nasal mucosa is pink over the septum and the middle and inferior turbinates appear larger and healthier. This appears to be an excellent result.

DISCUSSION.

Two years have passed since the first of these four cases was operated upon. It has appeared that in selected instances, bone grafts to the septum have been satisfactory in relieving the symptoms and signs of atrophic rhinitis. In the patients with good results, not only is there symptomatic improvement, but also the pale thin mucosa is changed to a more normal pink turgescent appearing membrane over the septum and turbinates. It is interesting that the narrowing of the nasal chamber by widening the septum with living bone causes the atrophic mucous membrane of the lateral nasal wall to approach a more normal color.

A properly prepared bony bed in the hard palate is a most essential requisite for success. The characteristics of the cancellous bone and the amount available also seem to be an important factor in securing a good result. Results with the use of iliac bone containing cartilage in the younger individual with chronic purulent sinusitis, or in the older person with osteoporotic bone indicate that at the present time we cannot expect a good result from this procedure alone.

Healthy and adequate iliac bone should be placed into the septum until nasal obstruction is obtained. Some resorption takes place during the first three- to four-month period before physiologically adequate airway is established. The insertion of cancellous bone into the septo-palatal area appears to be a reasonable and sound method for the relief of atrophic rhinitis. Further time will be required to tell whether the improvement gained will be permanent.

One case operated upon by the above technique and of less than two years' duration was not included in this report. To date, the result has been excellent.

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THE "FALSE-BING" PHENOMENON—SOME REMARKS ON THE THEORY OF BONE CONDUCTION.

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There can be no doubt that in all cases of pure conduction deafness, the bone conduction audiometric curve for the low frequencies is normal or very slightly below normal curve. All audiometricians agree with Professor Carhart's schematic curve[1] which shows a very smooth slope between 256 and 2048 cycles, where the maximum mean loss is about 15 db (see Fig. 1-a).

To begin with, we think it necessary to point out that Carhart's curve contradicts the presently accepted theory of bone conduction, which proceeds from the experimental findings of Barany and O. Bekesy. It is quite obvious that if, for the low frequencies, bone conduction is due primarily to the inertia of the ossicular chain, the fixation of the stapes should be translated on the audiogram by considerable loss up to about 1000 cycles. On the other hand, if, for the high frequencies, bone conduction is due primarily to the compression of the bony labyrinth, above 1000 cycles the effect of the compression of the semicircular canals should be translated by supranormal bone conduction. "We can suppose," writes I. Hirsh[2] "that if the stapes is fixed, as it is in otosclerosis, bone conduction will be better than normal, but primarily for the high frequencies when compression bone conduction plays the major role." In fact clinical otosclerosis is characterized by a bone conduction curve which according to theory is the exact contrary of what could be expected.

Let us consider a case of unilateral conduction deafness. We suppose for instance that the right ear is defective. with a bone-conduction curve normal, or perhaps slightly below

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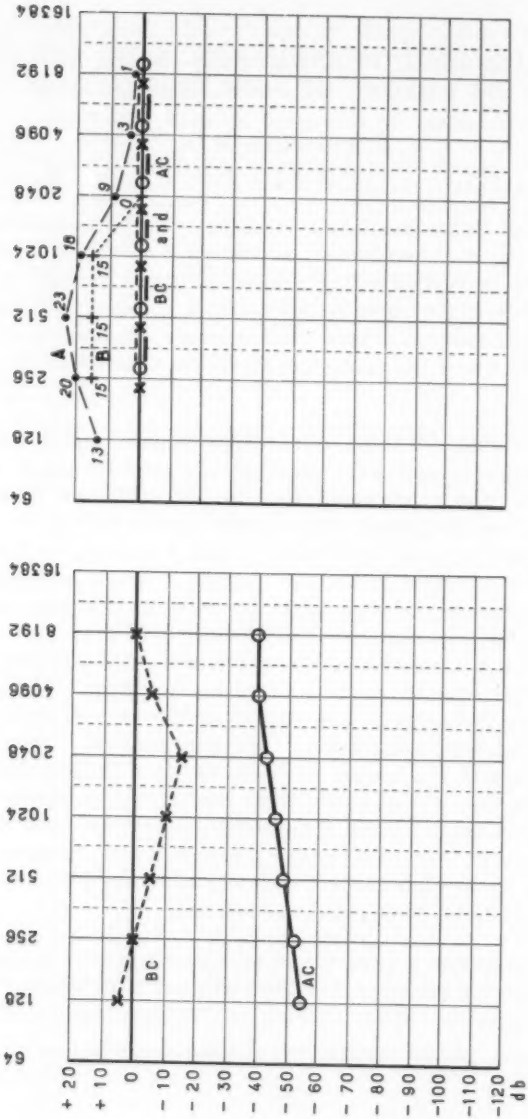

Fig. 1. a, . . . Schematic bone conduction audiometric curve in case of stapes fixation (according to Carhart). b, . . . Shift of bone conduction thresholds by occlusion of the Ear canal. A—wet cotton batting inserted (data from Sullivan, Gottlieb and Hodges). B-finger inserted.

normal on the low frequencies, and a left ear absolutely normal for the same frequencies. The Weber test shows that the bone conduction sounds are heard and localized in the right ear. Why should it be so, since at most both ears are normal as far as bone conduction is concerned? As far as we know, this has never been answered; in fact, one generally believes that if, in unilateral pure conduction deafness, the patient lateralizes bone conduction in the bad ear, it is because on that side, bone conduction is better than in the good ear; that is, better than normal.

It is easy to prove that this is not the case. To demonstrate, we must refer to the work of Sullivan, Gottlieb and Hodges on the occlusion shift for normal ears.[3] When the normal ear is occluded by means of wet cotton batting inserted in the canal, the bone conduction thresholds are shifted significantly above normal (see Fig. 1-b). We personally have no experience of this method of occlusion, but we have a wide one of the simpler method which consists in closing the ear with the finger, and, in such a case, the bone conduction shift is slightly below Sullivan's figures; it is about 15 db on the three frequencies, 256, 512 and 1024 cycles, the "0" index being between 30 and 59 db.

In a case of unilateral pure conduction deafness, if one occludes the good ear, it is evident that on that side bone conduction is significantly better than in the bad ear. The Weber test should then be lateralized in the good ear but, in fact, it is not. In all cases of unilateral pure conduction deafness of more than 30 db—and in 85 per cent of all cases—when one proceeds to close the good ear, the Weber remains lateralized to the bad ear. Moreover, and we consider this as very important, the occlusion of the good ear produces a significant shift of the bone conduction threshold of the bad ear, objectively as well as subjectively (see Fig. 2).

This, by analogy with the "False-Rinne negative" we have called the "False-Bing" phenomenon. It happens in all uni-

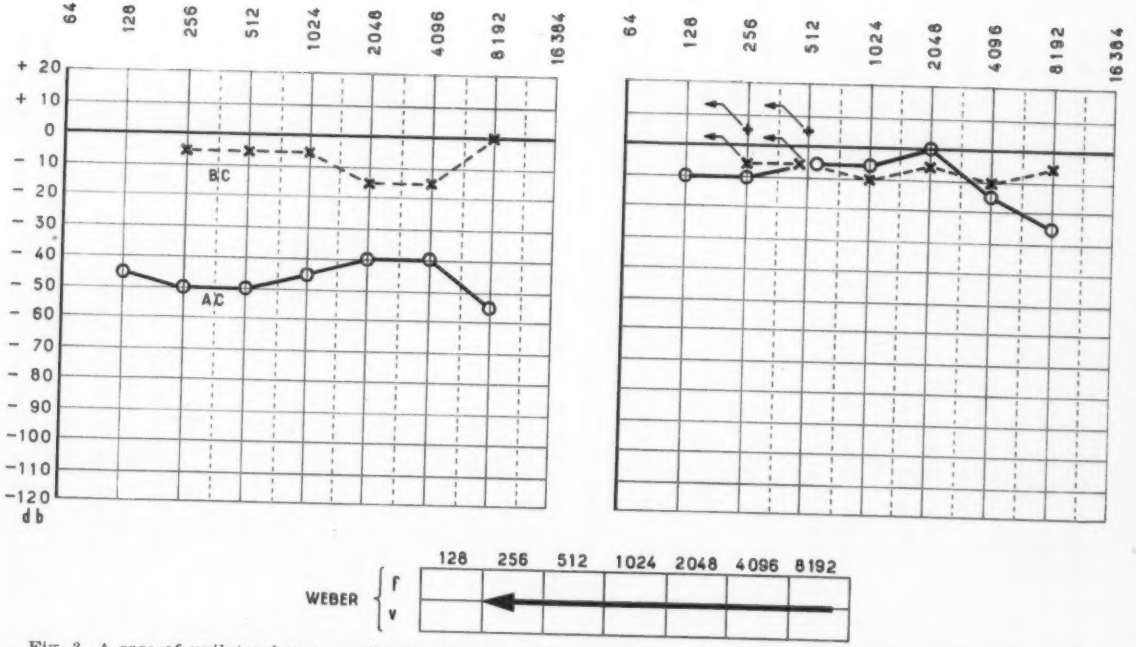

Fig. 2. A case of unilateral pure conductive deafness. Bone conduction threshold curves without any masking. The Weber is lateralized on the right side. As shown by the arrows on the frequencies 256 and 512 cycles, the occlusion of the left normal ear has produced a shift of 10 db in the bone conduction threshold, and the sensation is experienced by the patient in the defective right ear.

lateral pure conduction cases where the deficiency of the bad ear is above 30 db. One cannot always detect it for all the low frequencies; the probabilities of occurrence are:

256 c.	80 per cent.
512 c.	83 per cent.
1024 c.	43 per cent.
2048 c.	3 per cent.

Above 2048 c. the "False-Bing" phenomenon never occurs.

It should also be noted that this phenomenon occurs only in pathological cases. It does not happen when one tries to simulate a unilateral pure conduction deafness by occluding one ear with a finger. In that case the occlusion of the contralateral ear with a finger will easily and progressively reset the Weber in the center of the head.

Incidentally this experience shows that one should not consider that the Bing test (occlusion) and the Weber test are two different aspects of the same phenomenon. In Germany the terms applied to both tests make the confusion absolutely complete; in that country, the Bing test is called "Physiological Weber, because it seems that it was discovered at first by Weber and not by Bing, and the Weber test is called "Pathological Weber." The Bing test produces a significant shift of bone conduction threshold on the low frequencies, while the Weber test, in pathological cases, does not. The Bing test is, therefore, perfectly explained by O. Bekesy's hypothesis on the inertia of the ossicular chain, the effect of which is enhanced by the inertia of the head of the mandible vibrating in a closed canal. The Weber test is not explained by such a hypothesis.

What we have said above concerning the "False-Bing" phenomenon, shows that the Weber test remains unexplained: in unilateral pure conduction deafness, the Bone conducted low frequencies are heard in the bad ear, although we know for certain that on that ear bone conduction cannot be better than in the opposite good ear.

From the practical point of view, these findings have important bearings. It is utterly impossible in cases of unilateral pure conductive deafness to mask the bad ear in order to measure the bone conduction thresholds of the good ear, whatever may be the amount of white or saw tooth noise produced. Besides we may add that the present methods of calculation of the amount of noise necessary to mask an ear, do not take into account the fact that the bone conduction curve of the bad ear may be lower than the bone conduction curve of the good ear.

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HISTAMINIC CEPHALALGIA.* (SUICIDE HEADACHE).

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DEFINITION AND DESCRIPTION.

Histaminic cephalalgia is a two-phase unilateral, agonizing headache, occurring mostly at night or when the patient is relaxed, and results from rapid dilation of the external carotid artery. In typical cases, the pain areas follow the anatomical distribution of the artery. The attacks begin suddenly and are short, but frequently recur three or four times a night.

This headache is distinguished from migraine, which is a three-phase, unilateral headache beginning with vasoconstriction (when warning signs of impending attacks are present) followed by vasodilation and prolonged perivascular edema, which may persist for many hours or days.

I have called the syndrome "suicide headache" because so many of my patients have said that if they could not get relief, they would "end it all."

Patients will rave, scream, hit their heads against the wall; some will strike their heads with various objects in a futile attempt to deaden the pain. Since there is unilateral lacrimation during the severe pain, I have added another definition, "The headache of the raving people who weep with one eye only." This is descriptive of the extremely severe type of histaminic cephalalgia.

The following case history is illustrative:

H. H., age 46 years, a motor car executive, had terriffic unilateral night headaches for six years. Each attack was accompanied by unilateral lacrimation, nasal obstruction, and other symptoms. The attacks would

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last thirty minutes to two hours. He seldom escaped an evening or a night without one attack—sometimes three or four. He had noticed that his attacks came only when he was relaxed after work in the evening or they would awaken him out of a sound sleep. While sitting with his family in the evening watching television or listening to the radio, his children would become frightened when they saw his right eye begin to lacrimate. The children would dash out of the room or go to the neighbors until the screaming was over. He always appeared to be in a violent rage, but never injured anyone except himself. He seldom slept more than four hours; by morning, relief would come when he had to return to work, utterly exhausted.

He developed two methods of counter-attacks: 1. When the pain would start he would jump out of bed screaming, dash out of his home into a nearby park, dressed only in night clothing, even in severe winter. His wife would follow with slippers and robe. He would run and scream until the attack was over. The screaming and running seemed to lessen the severity of the attack. 2. He would hit himself on the head with a hammer in the vain hope of deadening the pain.

In the six years he had received 13 different diagnoses from 26 doctors in six states; several of the physicians were heads of departments in large teaching institutions. The most common diagnosis was a neurosis, always with the advice to have psychiatric treatment. Several times the condition was diagnosed as "sinusitus" and he had one unnecessary sinus operation. He was sent to a hosiptal one night where an exploratory intracranial operation was advised. Fortunately the operation was not performed, as the pain stopped suddenly. Other diagnoses were migraine, glaucoma, syphilis, pinched nerve, and bad teeth. Each physician consulted felt sure he understood the trouble, and various treatments, which never did any good were prescribed, always starting with morphine for the acute episode.

With the housing shortage following the war, he had great difficulty finding a place to live because of his nightly screaming episodes. He always tried to rent an apartment near a park or an athletic field, where he could run and scream without disturbing too many neighbors or attracting the attention of the police.

More than two months were needed to get this patient stabilized on the correct dosage of histamine, and other treatment, before he was relieved. During this period he was given a letter authorizing any physician, intern, resident or nurse to administer DHE_{45} intravenously. Frequently he would be relieved from the acute attack before the needle was out of his vein. His wife would drive him to the emergency room of the hospital; he could always drive home. He carried his own DHE.

ETIOLOGY.

The etiology of histaminic cephalalgia is unknown; however, I have developed a working hypothesis based only on clinical observations over more than ten years and with more than 100 patients. This hypothesis includes primary and secondary causes, and I will discuss the secondary first. The disease, I believe, is due to a secondary involvement of the au-

tonomic nervous system, the impulses from which are mediated through the great control center, the hypothalamus. The hypothalamus is the control tower that regulates the rhythmical contractions and dilations of all blood vessels, as well as the body rhythm.

The severe pain of histaminic cephalalgia is produced by an unusual dilation of the external carotid, which stretches all the surrounding sensory nerves and tissues. In the typical case the pain locations follow almost the exact areas supplied by the artery. The pain lasts as long as the artery remains expanded. The instant the artery regains its normal size the pain stops, which can occur in a fraction of a second, but more often there is a "fading out" period of a few minutes to half an hour.

There is little doubt that there is a release of histamine in the body just before the pain starts. Also, there is little doubt that there is an increased body sensitivity to histamine, as well as sensitivity to other allergens. I have been unable to prove to my own satisfaction that histamine sensitivity can be demonstrated by cutaneous testing to the extent that would be of any real diagnostic value. Others have contrary opinions.

It is assumed that there is a sudden release of histamine in the body followed immediately by dilation of the external carotid artery resulting in excruciating, unilateral head pain. It may be assumed, also, that a disturbed vascular rhythm is transmitted from the autonomic nervous system to the hypothalamus, the great regulator of vascular rhythms. Notwithstanding both of these physiologic assumptions, we still do not know the primary cause of the autonomic dysfunction —this disturbed rhythm.

Supported only by clinical observations, I believe that an imbalance or deficiency in electrolytic metabolism develops a nutritional or hormonal disturbance. If one would successfully manage this devastating disease he must be able to visualize the probable primary and secondary causes and combat them energetically for years.

SYMPTOMS AND DIAGNOSIS.

Certainly the diagnosis cannot be complete without an intensive, physiologic and analytic investigation of these phases. Women have said that their headaches, their allergies, even asthmatic bouts disappeared completely during pregnancy—especially during the seventh and eighth months when the estrogenic blood levels are the highest. Physicians have not given these voluntary statements the meticulous attention warranted by the physiologic facts observed. I am now making a vigorous and methodical investigation of all suspected hormonal disturbances—both estrogenic and androgenic.

Since the diagnosis is made almost entirely by symptoms, I will repeat the story told by a patient with typical histaminic cephalalgia:

> A young woman executive said on her first visit, "You are the last physician I am going to consult. I work hard all day and go home at night so exhausted I fall in bed, often without eating dinner. I am awakened out of sound sleep from one to five times a night with a pain so severe I have to jump out of bed. It is as though someone was plunging a knife into my right eye, my right temple, and the back of my neck. My head and eyes feel as though they would burst, the pain is so intense. The attacks last from 15 to 45 minutes; then all is well until the next bout begins in one or two hours. Of course, in the morning I am more exhausted than when I went to bed. I can't take it any longer!"

This intelligent young woman gave a classical history of a typical histaminic cephalalgia. When questioned further she described the characteristic unilateral nasal obstruction, lacrimation, flushing and, of course, redness and swelling of the right eye, all of which disappeared abruptly when the attack was over—only to recur when the next painful episode began.

Not all cases of histaminic cephalalgia are typical; not all patients give an intelligent history. Many will say, "I have terrible headaches," and leave it to the physician to find out as best he can the correct diagnosis. In a busy office or clinic the patient does not always get a chance to tell the whole story. Without the story a correct diagnosis is seldom made. It is easy to say, "You have a tension headache" or "a nervous headache" or "change your way of life," which the patients seldom can or will do. It is easy for an otolaryngol-

ogist to see a deviated septum, or perhaps a cloudy sinus, and otolaryngologists are often abetted in their misconception by roentgenologists who will find a slightly cloudy sinus or, as they frequently report, "thickened mucous membrane in a sinus suggesting an old chronic infection." This has, in the past, been sufficient evidence to start a march on a series of unnecessary and often mutilating surgical procedures on sinuses. Horton[1] says that over 40 per cent of his patients have had unnecessary surgical procedures.

In order to obviate these errors in my department at the Research Clinic, I insist that each patient who complains of headache be required to fill out a two-page detailed questionnaire. There are two fairly large blank spaces in which the patient may even elaborate on his symptoms. This, at least, makes him alert to the fact that I expect him to give me full details. I impress on him at the first consultation that one can seldom "see" a headache—it must be diagnosed by the symptoms. The one exception is the instance in which one is fortunate enough to see a patient in an attack. Since the pain usually occurs at night this is seldom possible. Nearly all patients will say they have sinus trouble because of the nasal symptoms which occur in histaminic cephalalgia. None has an empyema of the sinuses; many have a dysfunction of the nasal mucosa with profuse secretion in the nose and nasopharynx. This, *per se*, is not the cause of the pain, but it does provide a localizing symptomatic starting point.

MANAGEMENT AND TREATMENT.

Someone has said, "If I wished to show a student the difficulties of medical practice, I should give him a headache to treat." May I add to this sagacious remark that the physician must be prepared to expect these "difficulties" and to adjust himself to them. He must be unwilling to accept defeat. He must insist on ample time for observation and stabilization of therapy. The physician positively must not accept patients and be responsible for results unless the patient can be observed for at least one month. If the patient's progress is not satisfactory, the physician must be alert and willing

to change any or all the therapeutic procedures. In a number of instances I have changed the whole unfavorable outlook by restudying the hormonal status of the patient. I test the patient over a three-day period to determine the minimal effective dose of hormones. The physician must not accuse the patient of being psychotic because good results are not immediately obtained.

Fractional histamine is the most important part of the treatment and should be continued indefinitely, yet it does not by any means comprise the entire necessary therapeutic regime. The pain is produced by the vasodilating mechanism, with a disturbance of rhythm between vasodilation and vasoconstriction. Before the perplexing, dramatic and agonizing disease can be treated with any hope of success, certain fundamental physiologic and pharmacologic facts must be known and applied by the physician:

Epinephrine constricts extracranial vessels; does not constrict intracranial vessels. It may be tried in doses of 1 cc. of a 1:100,000 solution, intravenously, to relieve the acute attack.

DHE_{45} constricts extracranial and intracranial vessels and will relieve the acute attack in a few minutes. It must be given intravenously. Opiates will not give relief. A vasoconstrictor is the only effective remedy.

Histamine dilates both intracranial and extracranial vessels, arterioles, venules and capillaries.

Epinephrine and histamine are physiologic antagonists, except for their action on the intracranial vessels.

I know of no other disease that requires more intensive study and direction by the physician. I believe that there is a dysfunction of the autonomic nervous system, and probably a deficiency or imbalance of many of the body's most important physiologic processes. Horton[1] says, "There is no short cut in the treatment of headache, and the so-called sure cures usually prove disappointing." He advises histamine desensitization as follows: He gives subcutaneously twice daily—8 a.m. and 4 p.m.—Histamine Diphosphate 0.275 mg. per cc.

He starts with 0.10 cc.—0.15 c.c. the first day; second day, 0.20 cc.—0.25 cc. increasing at this rate until 0.50 cc. are given. He says that he has never had to administer more than 1 cc. My own experience has convinced me that much smaller amounts of the drug are effective. If I give larger doses or give it intravenously, even in the weakest dilutions, which is sometimes necessary, I am almost certain to incite an acute attack. I proceed on the principle of hyposensitization. I start with 0.10 cc. twice daily. If I obtain good results, the dose is never increased. I treat all my allergic patients on the same principle; namely, the smallest possible dose that will get results. I treat my allergic patients only when they are in trouble, while the histaminic cephalalgia patient must be treated over a period varying from months to years, depending upon his response. I frequently increase the interval of the histamine injections and prescribe it three times a day; however, this is seldom necessary. Frequently, I find that 0.05 cc. or 0.10 cc. will be ample once a day, or every other day. As stated above it is necessary in the very refractory case to give histamine by the intravenous method in the very weak dilutions (1:7,500,000); also, I see that all deficiencies or imbalances are corrected. In the beginning I give each patient large doses of vitamins and minerals. I usually prescribe some form of potassium.

I warn each patient that he must not discontinue any medication or break any rules of the regime until he has been symptom free for one year. Patients are trained in the self-injection of histamine. Of course, it would be impossible and impractical for them to get it any other way. The following instruction card is given on the first visit.

Instructions for Self Injections of Histamine Diphosphate

Histamine Diphosphate 0.275 (Abbott), must be taken subcutaneously (under the skin) twice a day. This is subject to change only by the doctor and will be done according to your improvement. Start 0.10 twice daily and continue twice daily until instructed otherwise. Since each individual case is different it may be necessary for the doctor to advance the

dosage to 0.15, 0.25, or 0.30 two (2) or three (3) times a day. We seldom find it necessary to go above the dosage just mentioned. It is our object to keep the dosage as small as possible. Be sure to inform us each time you return to the office, exactly how much pain you have had. When you go two weeks without severe pain, we usually reduce the dosage or increase the interval between doses. If you notice flushing of your face after taking Histamine please report this to us.

Steps for Filling Syringe:

Wipe off top of bottle with alcohol sponge or cotton. Pull back on the plunger of the syringe and inject some air into the bottle being sure to have the syringe and plunger securely fitted into the hand. After injecting the air, draw back more solution into the syringe than needed, holding the bottle up straight. This is necessary in order to get any air bubbles out of the syringe, back into the bottle, as you cannot possibly have the accurate dosage required when there is air in the syringe.

With the plunger on the exact mark of dosage, remove the syringe from the bottle. May we say again, PLEASE HOLD PLUNGER AND SYRINGE SECURELY.

Preparation of the Skin for Injection:

This is more easily given in the upper leg.

Wipe the area of injection with an alcohol sponge before giving yourself the injection.

With your free hand hold up on the flesh securely and stick the needle straight into the flesh, push the plunger down into the syringe, and then remove quickly, wiping the area once again with an alcohol sponge.

If DHE_{45} or other medication is to be given use the same technique in handling the syringe.

PROGNOSIS.

An immediate cure cannot be promised. I prefer to use the word "arrestability." Seldom is there failure to arrest the condition. I have a large number of patients with a five-year arrestability—some much longer.

Only recently I had one of my five-year "arrestables" return with his old trouble, and I had to start therapy again.

This painful, disabling, discouraging disease, frequently leads to serious despondency. The patient should be informed in the beginning just what the probable outlook will be; what he can expect and what not to expect. The disease is not hopeless where there is a cooperative patient and competent management.

CONCLUSIONS AND COMMENTS.

The true etiology of histaminic cephalalgia is unknown. I postulate a disturbed vascular rhythm causing a prolonged dilation of the external carotid artery. This dilation stretches all of the sensory nerves and tissues throughout the entire anatomical distribution of this artery, producing a most excruciating pain.

I also postulate that there is a dysfunction of the autonomic nervous system as it mediates nerve fibers to the hypothalamus, which is the control center for vascular rhythm, both contraction and dilation. The primary cause may be a deficiency or imbalance of electrolytic metabolism; therefore, nutritional and hormonal deficiencies and imbalances are carefully appraised.

The diagnosis is made on symptomatology alone.

The following symptoms are pathognomonic of the typical histaminic cephalalgia:

1. Severe unilateral headache occurring mostly at night, awakening the patient out of a sound sleep.
2. The attacks are of short duration; come on suddenly and end abruptly. They usually last from 30 minutes to two hours. They are not accompanied by nausea or vomiting.

3. The patient cannot stay in bed with this type of head pain.

4. There is nasal stuffiness and profuse lacrimation, redness and swelling of the eye on the affected side.

5. The external and internal carotid arteries are both tender during the acute attack. Many patients find that compression over these areas will give some temporary relief.

This paper describes one of the most painful syndromes known. A wrong diagnosis is the rule, not the exception, even by most experienced physicians.

This presentation offers nothing that is new except the low dosage of histamine during the desensitization period, and minute detailed attention to deficiencies of all sorts, including hormones.

To Dr. Bayard T. Horton should go great honor from the medical profession all over the world for his splendid clinical research on the disease that bears his name—"Horton's Syndrome."

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1110 Professional Bldg.

A COMPARISON OF HEXYLCAINE WITH COCAINE AS APPLIED TO THE PRACTICE OF OTOLARYNGOLOGY.

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In a previous publication[1] a comparison was made of tetracaine and cocaine, with emphasis on their uses in the practice of otolaryngology. Similar methods have now been applied to a study of hexylcaine. Hexylcaine is distributed under the name "Cyclaine" by Sharp and Dohme, Inc.

In 1947, after extensive pharmacological studies, Beyer, Latven and Freiburger concluded that the toxicity of hexylcaine lay between procaine and cocaine.

Method. Animal tests:

Guinea pigs weighing approximately 1 kilo were used. A small amount (0.5 cc. ½ per cent procaine hydrochloride) was injected intracutaneously in the midline of the neck from the suprasternal notch to the submental area. The trachea was then exposed and carefully dissected free over a distance of 2 cm. through a longitudinal incision. A 5 per cent solution of hexylcaine was instilled drop by drop into the intact trachea, and the amount required to produce convulsions and death was noted. Twitching could be produced after 0.2-0.3 cc. had been injected, but gross terminal convulsions and death could not be produced by this method without drowning the animal. If the injection was discontinued for 3-4 minutes, the animals would make an almost complete recovery (i.e. twitching would cease, respirations would return to normal rate and rhythm).

RESULTS.

With each animal the total amount injected exceeded 1.0 cc. without death. After injecting 0.75 to 1.25 cc. the animals

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were unconscious, but only twitching and inconstant minor convulsions were present. Death could be produced rapidly by the further injection of 0.2 cc. intravenously.

From the above results, cocaine would appear to be at least twice as toxic as hexylcaine.

Absorption from the tracheal mucosa was rapid in each case and, as has been mentioned, the recovery was moderately rapid when injection was discontinued.

Clinical experiments. Intranasal tests:

Following the procedure developed previously, the potency of hexylcaine was compared with that of cocaine by testing and comparing 5 per cent solutions of each drug when applied to the nasal mucosa of patients visiting the Out-patient Department of the Toronto General Hospital. Each patient was examined prior to testing, and particular attention was paid to the condition of the nasal mucosa of each individual. Only those patients with apparently normal mucous membrane were used. Eight men and four women, ranging in age from 18-60 were used.

The procedure was as follows: Half inch squares of blotting paper impregnated with constant amounts of 5 per cent cocaine and 5 per cent hexylcaine hydrochloride were inserted into the nose and applied to the nasal septum after first determining that normal sensation to pin prick was present. The cocaine was applied to one side and the hexylcaine to the other side of the nasal septum. Sensation was tested at one minute intervals until the patient declared that the sharp pin prick sensation was absent. This time was called the time of onset. The blotting paper was then removed and the time for return of normal sensation was noted and referred to as time of duration.

RESULTS—*See Table 1.*

No significant difference could be seen in times of onset with the two anesthetics but there was a constant difference in time of duration, hexylcaine being slightly longer than cocaine. This difference was from one to two minutes.

TABLE 1.

POTENCY STUDIES
Solutions applied to nasal mucosa of septum

Cocaine 5%		Cyclaine 5%	
Onset in min.	Duration in min.	Onset in min.	Duration in min.
3	5	2	7
3	4	4	5
3	7	2	9
2½	2	2½	4
2	2	2	6
2½	6	2½	11
2½	5	2	8
1	8	1	10
2	6	2	9
2½	8	3	8
1	5	1	7
2	6	2	8

Clinical Trials. Endoscopic examination.

In the past, cocaine hydrochloride has been the anesthetic most often used for endoscopic procedure at the Toronto General Hospital, except for a one- to two-year period when tetracaine was used widely. The concentration of cocaine most often used was a 5 per cent solution. It was decided to try 5 per cent hexylcaine as the local anesthetic agent, using as a basis of comparison past experience with cocaine.

Twenty patients were used. The pre-operative sedative in each case was the same as that administered when cocaine was used (i.e. morphine 1/6 gr.-1/4 gr., atropine 1/150 gr., nembutal 1½ gr. The method adopted for applying the anesthetic was identical with that used with 5 per cent cocaine and was as follows: The pharynx was sprayed with approximately 2 cc. of the solution after which both piriform fossae and the supra glottic area were swabbed with cotton pledgets moistened with 5 per cent hexylcaine. Two cc. of solution was then dropped between the vocal cords into the trachea. The total amount used did not exceed 5 cc.

The amount of coughing observed during the endoscopic procedure was the main criterion for comparison. The results were tabulated as good (i.e. as good as the best results pre-

viously obtained with 5 per cent cocaine), fair, and poor. In 18 of the 20 cases epinephrine in a final concentration of 1:50,000 was used with the anesthetic solution. In two cases no epinephrine was used.

RESULTS.

Fourteen of the 18 cases showed good anesthesia. Four cases were classified as fair, but it was felt that these cases would have been only fair with any topical anesthetic. All four were seen to have excess mucus, generalized thickening of the mucous membrane, or generalized acute inflammation of the mucous membrane.

The two cases which received hexylcaine without epinephrine were similar in every respect to those who did receive epinephrine in the anesthetic solution.

No untoward reactions were noted. There was no evidence of twitching or convulsions, and there was no visible damage to the tracheal or bronchial mucosa.

It was felt that hexylcaine in a 5 per cent solution produced anesthesia as satisfactory as might be expected with 5 per cent cocaine.

Hydrolysis in human serum.

The hydrolysis of hexylcaine in human serum was observed over a period of time, using the Beckman quartz spectrophotometer. Moderately purified human serum esterase was prepared from outdated blood from a blood bank. Studies were done at room temperature and results with hexylcaine were compared with procaine hydrolysis performed in a similar manner. Detailed description of the method of studying the hydrolysis is omitted from this paper and the reader is referred to a paper by Kalow.[2]

It was found that hexlycaine was broken down slowly in human serum, four to eight times more slowly than procaine. In 100 minutes 3.5 micrograms of procaine HC1 as compared to 0.8 to 0.4 micrograms of hexylcaine HC1 were destroyed

by the same preparation of human serum estarase. This finding contrasted with the observation made during toxicity studies with the experimental animal where it could be safely assumed that hexylcaine was broken down rapidly, allowing the animal to recover if the injections of the drug were interrupted for a few minutes. This finding serves to emphasize what is seen frequently, namely, that animal toxicity studies in particular cannot be safely applied to the human subject.

It is known that cocaine undergoes almost negligible hydrolysis in human serum, and the rate of hydrolysis of procaine is considered to be moderately rapid.

SUMMARY.

1. Toxicity studies using guinea pigs are presented and it is seen that cocaine is at least twice as toxic as hexlycaine.

2. Potency studies using humans are presented. Hexylcaine is found to produce anesthesia at least as prolonged as with an equal amount of cocaine.

Hexylcaine was found to be as efficient as cocaine in the production of local anesthesia for the performance of endoscopic procedures.

3. The hydrolysis of hexylcaine by human serum esterase was found to be four to eight times slower than hydrolysis of procaine.

CONCLUSIONS.

Hexylcaine appears to be as efficient a local anesthetic as cocaine for endoscopic procedures and appears to have a greater margin of safety than cocaine.

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METASTASIS OF MALIGNANT TUMORS TO THE LARYNX.

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The purpose of this article is to note the unusual occurrence of malignant tumors metastizing to the larynx. The lymphomata group are not included.

A review of the world literature from 1916 through 1951 reveals five authentic cases of tumor metastasis to the larynx.

CASE REPORTS.

Case 1: In 1924 Turner reported the first well documented case of a renal cell carcinoma metastizing to the larynx. A seventy-year-old white male was admitted to the hospital with a chief complaint of hoarseness of three months' duration. A smooth pale subglottic swelling directly beneath the right true cord was noted on examination. At autopsy this mass was found to be a metastasis from a renal cell carcinoma of the right kidney.[1]

Case 2: In 1931 Oppikoder reported the case of a fifty-year-old white male who entered the clinic with a chief complaint of dysphagia of two months' duration. A one centimeter, nodular, blue-red mass involving the left half of the epiglottis was found on examination. The tumor was locally excised and thought to be either a metastasis from a renal cell carcinoma, a thyroid carcinoma or a sarcoma. A review of the patient's history revealed that a left nephrectomy for a renal cell carcinoma had been done six years prior to the discovery of the epiglottic lesion. No further tumor spread was noted for one and one-half years following local removal of the epiglottc mass.[2]

Case 3: In 1941 Havens and Parkhill reported the case of a forty-year-old white male who entered the clinic with a chief complaint of throat irritation of one year's duration. A malignant appearing dark brown mass was seen arising from the right arytenoid cartilage. Pathological examination of the tumor revealed a diagnosis of malignant melanoma. Other widespread metastasis presumably from a primary skin lesion were present.[3]

Case 4: In 1951 Fisher and Odess reported the case of a sixty-three-year-old white female who entered the hospital with a chief complaint of hoarseness of eight months' duration. On examination a round, smooth, dark colored tumor involving the right vocal cord was seen.

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A malignant melanoma had been removed from the right shoulder three years previously. A laryngectomy was done, and a diagnosis of malignant melanoma of the larynx was made.[4]

Case 5: In 1952 one of us (J. R. L.) reported the case of a sixty-eight-year-old white male who entered the hospital wth a chief complaint of hoarseness of five months' duration. A greyish-pink, grape-like tumor 6 mm. in diameter was seen attached to the left true cord. This was removed and found to be a malignant melanoma. The probable primary lesion was found on the skin of the right shoulder. Other wide spread metastasis soon became apparent.[5]

Several cases of lesions involving the larynx which were presumably metastatic growths from renal cell carcinomas are present in the literature but were not proven by renal examination.[1,2,6,7]

DISCUSSION.

Two theories are generally accepted to account for the scarcity of tumor metastasis to certain regions:

(1) Some tissues present less favorable environment for tumor growth.

(2) Failure of tumor emboli to reach certain[8] regions in sufficient number for growth.

In support of the latter, Coman et al., in experimental work with sarcoma in rabbits, found that tumor metastasis would develop in any organ that received viable tumor emboli in sufficient numbers. Muscle metastasis developed readily after injection of emboli into the left heart chamber.[9]

Tumor emboli are probably arrested in the first capillary network encountered. Emboli liberated in the systemic veins are caught in the lungs, those entering the portal may lodge in the liver and those entering the pulmonary veins may presumably be deposited in any peripheral organ.[8]

Batson pointed out the possible role of the vertebral veins in the spread of metastasis by retrograde flow thus yielding systemic spread without passing through the lungs.[10]

The location of the larynx is such that it is probably reached by relatively few tumor emboli. Only those tumors which metastasize widely and profusely would presumably be encountered as metastatic to the larynx. Renal cell carcinoma and malignant melanoma fall into this category.

SUMMARY.

A review of the literature reveals two acceptable cases of metastasis of renal cell carcinoma to the larynx and three cases of metastasis of malignant melanoma to the larynx. It is believed that the scarcity of metastatic lesions involving the larynx is due to the scarcity of tumor emboli reaching the larynx.

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HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

January 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A-300; 1 transistor, 2 tubes, and 2 batteries.
Manufacturer: Dictograph Products., Inc., 95-25 149th St., Jamaica 35, N. Y.

Audivox, Model 71; all transistor.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Maico Transist-Ear, Model O; 3 transistors and 1 battery.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis, 1.

Otarion Model C-15; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Sonotone Model 1010; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Telex, Inc., Telex Park, St. Paul, 1.

Zenith Model Royal-T; 3 transistors, and 1 battery. Zenith Model Super Royal-T; 3 transistors, and 1 battery.
Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
Vice-President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Boston, Mass., May 23-24, 1954.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Gordon F. Harkness, Davenport, Iowa.
First Vice-President: Claude C. Cody, Houston, Tex.
Second Vice-President: Daniel S. Cunning, New York, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Boston, Mass., May 28-29, 1954.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass.
President-Elect: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Boston, Mass., May 25-27, 1954. (Mornings only.)

SECTION MEETINGS.

Eastern Section: Friday, Jan. 8, 1954, New York City, Waldorf Astoria Hotel.
Council Meeting: Saturday, Jan. 9, 1954, New York City, Waldorf Astoria Hotel.
Southern Section: Saturday, Jan. 16, 1954, Louisville, Ky., Brown Hotel.
Middle Section: Monday, Jan. 18, 1954, St. Louis, Mo., Park Plaza Hotel.
Western Section: Saturday, Feb. 6, 1954, Portland, Ore., University of Oregon Medical School.

(In Portland, room reservations may be made at Heathman Hotels).

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Dean Lierle, Iowa City, Iowa.
Vice-Chairman: Dr. Fred W. Dixon, Rose Bldg., Cleveland, Ohio.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.
Meeting: San Francisco, Calif., June 21-25, 1954.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago 11, Ill.
President-Elect: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Waldorf-Astoria, New York City, Sept. 19-24, 1954.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Statler Hotel, Boston, Mass., May 17-22, 1954.
Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, 1100 No. Charles St., Baltimore 1, Md.
Secretary: Dr. F. Johnson Putney, 255 So. 17th St., Philadelphia (3) Pa.
Meeting: Statler Hotel, Boston, Mass. (Afternoons) May 25-26, 1954.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis, Ind.
President-Elect: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Waldorf-Astoria, New York City, Sept., 1954.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonso, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: Feb. 28 to Mar. 4, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Norris J. Heckel, Chicago, Ill.
President-Elect: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting: Chicago, Ill., Sept. 22-24, 1954.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Peter N Pastore, Richmond, Va.
President-Elect: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
Vice-President: Dr. H. L. Mitchell, Lexington, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Orwyn H. Ellis, M.D.
Secretary-Treasurer: Harold Owens, M.D.
Chairman of Section on Ophthalmology: Robert A. Norene, M.D.
Secretary of Section on Ophthalmology: Sol Rome, M.D.
Chairman of Section on Otolaryngology: Leland R. House, M.D.
Secretary of Section on Otolaryngology: Max E. Pohlman, M.D.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to June, inclusive—Otolaryngology Section; 6:00 P.M., first Thursday of each month from September to June, inclusive—Ophthalmology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Fleit, 66 Park Ave., New York (16), N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. William Banks Anderson, Durham, N. Car.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Harry P. Schenck.
Vice-President: Dr. William J. Hitschler.
Treasurer: Dr. Chevalier L. Jackson.
Secretary: Dr. John J. O'Keefe.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. M. Valentine Miller, Dr. Charles E. Towson, Dr. Thomas F. Furlong, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto, Ontario.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place:
Time:

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. Alberto P. Haedo.
Vice-Presidente: Dr. V. R. Carri.
Secretario: Dr. Renoto Segre.
Pro-Secretario: Dr. Carlos A. Gutierrez.
Tesorero: Dr. J. M. Tato.
Pro-Tesorero: Dr. Norberto Von Soubiron.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

THIRD LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Franze Conde Jahn, Caracas.
Secretary: Dr. Victorino Marquez Reveron, Caracas.
Meeting: Caracas, Venezuela, Feb. 21-25, 1954.

BOOK REVIEW.

Respiratory Diseases and Allergy. New Method of Approach. By Josef S. Smul, M.D., Fellow, National Gastro-Ent. Association; Formerly Vice President, Manhattan Roentgen Ray Society; Association of Gastro-Ent; Beth David Hospital; Clinical Assistant, Physicians Beth Israel Hospital. Eighty Pages with Index. New York: Medical Library Company, 232 East 15th Str. 1953. Price $2.75.

A note book is of not much value to anyone other than the author. This is a note book. It contains nothing new except some terms such as Respirallergy, Laryngallergy, etc., without which we would be much better off!

The book discusses respiratory diseases in some 72 pages; has an Index and one reference! T. E. W.

ANNOUNCEMENT.

The Annual Convention of the Oklahoma City Academy of Ophthalmology and Otolaryngology with the Postgraduate Department of the University of Oklahoma Medical School will be held March 26, 1954. Guest speakers will be Paul A. Chandler, M.D., Harvard University, Ophthalmologist, Boston, Massachusetts, and Theo. E. Walsh, M.D., Washington University, Otolaryngologist, St. Louis, Missouri.

Registration fee of $12.00 includes registration, social hour, and dinner. The program will include question and answer periods.

1954 CONGRESS OF THE SOCIETA ITALIANA DI OTORINOLARINGOIATRIA.

For the next Congress of the Societá Italiana di Otorinolaringoiatria in 1954 the following report subject has been chosen: "Congenital Malformations in the field of Oto-rhinolaryngology."

The authors who have published their researches on this subject are kindly asked to communicate the titles to Dott. Tomaso Marullo, Clinica Otorinolaringoiatria, Universitá, Roma, or, if possible, to send him a presentation copy.

BRONCHOESOPHAGOLOGY COURSE.

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period, March 22 through April 3, 1954. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

PROCEEDINGS OF BRITISH ASSOCIATION OF OTOLARYNGOLOGISTS.

The British Association of Otolaryngologists has copies of the Proceedings of the 4th International Congress of Otorhinolaryngology for sale at Five Guineas each.

These papers are numerous and the publication is well produced.

Application for copies should be made to The Secretary, British Medical Journal, B.M.A. House, Tavistock Square, London, W.C.1.

KENFIELD MEMORIAL AWARD.

Competition for the Kenfield Memorial Scholarship, awarded annually by the American Hearing Society to a prospective teacher of lipreading, will open March 1. Application blanks may be obtained by writing to the society's national headquarters, 817 - 14th St., N.W., Washington 5, D. C.

Deadline for returning completed applications is May 1. They are to be mailed to Mrs. Eleanor C. Ronnei, care of the New York League for the Hard of Hearing, 480 Lexington Ave., New York 17, N. Y. The winner will be announced during National Hearing Week, May 2 - 8.

Winner of the annual award is entitled to take a teacher training course in lipreading from any school or university in the United States acceptable to the Teachers Committee. The scholarship is to be used within one year from the date of award.

A satisfactory applicant for the award must be a well adjusted individual with a pleasing personality, legible lips, good speech and voice and no unpleasant mannerisms. Graduation from college with a major in education, psychology and/or speech is a requirement.

Rules for competition state that an applicant shall plan to teach lipreading with or without other types of speech or hearing therapy.

CONTENTS